CIVIL VETERINARY DEPARTMENT

LEDGER SERIES No.

BENGAL SECRETARIAT LIBRARY

MAIN HEAD . . STOCK DISEASE.

SUB-HEAD . . CONTAGIOUS DISEASE.

SUBJECT:

RINDERPEST—CATTLE PLAGUE.

1894.

CALCUTTA:
OFFICE OF THE SUPERINTENDENT OF GOVERNMENT PRINTING, INDIA.
1894.

CALCUTTA
GOVERNMENT OF INDIA CENTRAL PRINTING OFFICE,
8, HASTINGS STREET

PREFACE.

THE enclosed article, which has been collected from various sources, but chiefly from M. Galtier's "Maladies Contagieuses," is forwarded for information. M. Galtier's article is from his last edition. The following works have also been consulted: Walley's "Four Bovine Scourges," Fleming's "Sanitary Science," "Report of the Indian Cattle Plague Commission," Gunn's "Cattle Diseases in the Central Provinces," Thacker's "Reports to the Board of Revenue, Madras," and my own "Reports to the Government of the Punjab." The article is circulated interleaved with a request that any additions or corrections may be made on the blank sheets. Information on any disputed or doubtful point will be acceptable.

HENRY T. PEASE, F.R.G.S., F.Z.S.,
Asst. to the Inspector-General,
Civil Veterinary Dept.

MEERUT, 1894.

BENGAL SECRETARIAT LIBRARY

RINDERPEST—CATTLE PLAGUE—TYPHUS.

Rinderpest is undoubtedly the most widespread and important of all the diseases of cattle which are to be met with in India and Burma. There are certain districts where the malady never ceases from one year's end to another, but drags on its course, sometimes severe, at others apparently dying out, but in reality only flickering and ready to burst forth with renewed vigor at the first favourable opportunity. No just estimate can be formed of the enormous numbers of animals which annually perish from the disorder, as we have no accurate reports which may guide us in so doing; but from my own personal experience I can say that deaths from all other diseases to which bovine flesh is heir, taken together, will not nearly equal those which it causes. It is the custom in many parts of the Indian Empire to make light of the damage done by epizootic diseases and to cloak them over as much as possible; but such a policy can only emanate from weakness and a desire to evade instead of facing and overcoming the obvious difficulties which arise when the suppression or limitation of their spread has to be undertaken. No one, I am sure, has a more accurate appreciation of what these difficulties are than I have; but at the same time it is clear to me that much can be done to limit spread, if they be faced and proper measures adopted. It therefore behoves us to study this malady and to make ourselves acquainted with what is known at the present time regarding it.

Definition and general considerations.—Rinderpest is an epidemic, contagious, virulent general disease, infectious and very easily transmissible from a diseased to a healthy animal. It is characterized by groups of symptoms, which, although differing greatly in different countries, enable us to diagnose the malady as a rule. Amongst the more characteristic symptoms of this malady may be noted a low typhoid form of fever, ushered in by trembling, shivering, or rigors, and often also by muscular twitchings of certain groups of muscles. A discharge from the eyes and nose, first watery and soon becoming mucous; constipation at first, but in a few days followed by diarrhœa, and later fatal dysentery. Congestion of the skin, and in numerous cases in this country the eruption of pustules and papules on it, which has led some observers to consider the disease to be of a varioious nature. These are the

general symptoms observed in India. In some cases here, however, and invariably in Europe, other symptoms are found, which, when they appear, are considered to be pathognomonic of the malady, and these are discoloration of the mucous membrane of the mouth, followed by desquamation of its epithelium, leading to the formation of extensive characteristic sores covered by bran-like flakes of epithelium, and also congestion of the vaginal mucous membrane in the cow. The post mortem lesions of Rinderpest are more characteristic than the symptoms, and the more important of them are to be found in the digestive organs, especially at the pylorus and intestines, and also on the skin. These lesions consist of congestions, ecchymoses, infiltrations, swelling and desquamation of the epithelium, the presence of pustules and the like.

With regard to the *vera causa*, the virus of this disease, the researches which have been made up till the present time have not evolved definitely what it may be; but it appears probable that the latest enquiries in Russia have led to its discovery in the form of a micrococcus. Whatever the intimate cause of the virulence may be, it is undoubtedly generally disseminated throughout the whole of the organs and tissues of the body and exists in the blood, all the fluids and solids and the discharges coming from the sick animal.

Rinderpest, as I have said before, is always present in India, and it is, so far as my experience has been, never absent from the Himalayas, whither it has probably been introduced from China. It is a curious fact that all countries bordering on China are subject to the never-ceasing ravages of the plague, and that such has been the case for an exceedingly long time may be probably inferred from the fact that its virulence has become much decreased, as is the case with other diseases—syphilis for instance. We are in India, in this respect, in the same position as the Russians who have Rinderpest permanently located in their country. It is a fact which has been recognized for a very long time that Rinderpest occurring in countries where it is enzootic is much less severe than it is in countries to which it is newly introduced, and hence it is much less severe in India and in Russia than in England, Holland, Switzerland, Italy, and France, where it only occurs as the result of introduction from without of either diseased animals or material bearing the virus. It varies very considerably in different parts of India, and so marked has this variation been that I have been led to attribute it to the influence of the climate, and it appears probable that the great heat of the plains has some attenuating effect on the *materies morbi*. I have seen the disease in the Himalayas clear out the cattle of a small State, leaving less than a dozen animals alive in the whole of it, and have traced its spread down through over thirty villages, in which three fourths of the cattle have perished. It is no uncommon thing in the Himalayas for whole villages to be left destitute of cattle. The climate

of these parts is cool or cold and more or less moist. In the plain districts of the Punjab, on the other hand, the disease is of a much milder type as a rule, and 60 to 65 % of recoveries may sometimes be observed under treatment. These parts have an exceedingly dry and hot climate in the hot weather when the disease usually appears, and it seems reasonable to infer from the above facts that there may be some attenuation brought about by the heat and dessication. In India, as in Russia, we often get the disease in what has been termed the "benignant" form. It may be so slight as to pass unnoticed, there being no symptoms beyond slight fever and looseness of the bowels, and it is to this form that is alluded to as existing when the disease has apparently died out, but is in reality only flickering, and ready to burst forth again at the first favourable opportunity. The same occurs in Steppe cattle, and it has happened that Russian oxen imported to occidental countries have brought typhus with them when they have not been even suspected to be suffering from the disease.

It appears from the literature on the subject that Rinderpest originated in the extreme orient China, and has been spread by nomads and traders. The Huns are credited with the introduction of the disease into Europe.

Symptoms.—It will be well to give the symptoms of this disease as they are usually seen in Europe, and to compare them with those which are commonly seen in India, because in certain cases, seasons, and localities, the symptoms which characterize the malady in Europe appear in this country. It must be admitted, however, that the differences are often so great that one well acquainted with the European form of the disease would not recognize the Indian form. Although Rinderpest generally attacks bovine animals, its invasion is not specially confined to these; it may be observed in all ruminants, even in wild ones. I have seen it in the Kakar or barking deer in the hills, and large numbers perished from it in 1891 beyond Simla. It is particularly fatal in the camel. It has been seen in the Gaur or Indian bison, so called; in the wild buffalo, Tsain, etc. It is also found in certain porcines, the pig itself being subject to it. Victor D'Azyr thought that it could be transmitted to the horse, dog, and cat, but this is a mistake. In order to make ourselves thoroughly acquainted with the physiognomy of the affection, it is necessary to study it closely from its very commencement in several individuals. It becomes evident, then, that it may present itself in various forms which vary from the influence of a number of circumstances, with the country, climate, season, the hygienic conditions under which the animals are kept, the race of animals affected, etc., etc. It has been noticed to be more severe in soft lymphatic animals than in robust ones used to the open air; more severe in animals badly fed and cared for than in those well fed and cared for; it is more severe in winter,

when the animals are shut up in stalls, than in the summer; less severe in animals existing under good hygienic conditions than in those badly housed and fed; more severe in the hills than in the plains of India, etc., etc.

Rinderpest, like other contagious maladies, does not declare itself suddenly after the introduction of its germ into the organism, but there is the usual period of incubation, the duration of which is variable, but generally short: this period is ordinarily from 4 to 21 days, but the medium duration is from 6 to 10 days or from 5 to 9 days. In several cases inoculated by me, there was fever after from the 4th to the 6th day following the operation; but this is too small an experience to place any weight upon.

The first symptom of the existence of Rinderpest is a rise of the internal temperature, often preceded or accompanied by shivering or rigor. Although this sign of itself, occurring as it does in a host of other affections, has no precise diagnostic value, it assumes one when the disease already exists in the village or on the farm, and it permits us at least to suspect the disease if not to certainly affirm its existence, and allows of our taking measures to prevent the animal in which it is noted becoming a new focus of the disease.

The animal temperature rises to 102° F. or 105° F., or even in some cases to 106° F. This elevation may occur in from 36 to 48 hours after inoculation, and often exists for two or three days before any other symptom is noted. It is more or less pronounced according as the disease is going to be more or less severe. In India we must not be misled by the great variation which exists in the temperatures of animals at different parts of the country. As a rule the temperature increases with the heat and decreases with the cold—at least such has been my experience.

In cases which assume an adynamic character, or a typhoid form, the rise of temperature may be so slight as to escape notice.

The disease also announces itself by other general symptoms which, although not pathognomonic, aid greatly in diagnosis. After the lapse of a day or two following the rise of temperature, we note dullness, prostration and weakness; the animal hangs its head, the back is arched, the general sensibility is exaggerated, more particularly that of the dorsal region; the skin is hotter than normal, dry and often congested, especially in India; the hair is turned or erect; the attitude denotes suffering, locomotion difficult and gait tottering; debility rapidly shows itself.

The appetite early becomes in the majority of cases diminished, capricious, or absent, although cases have occurred to my knowledge where the patient has fed till the end in India. Rumination is of shorter duration, irregular, or absent. The respirations are rapidly increased by the slightest exertion. The circulation is generally accelerated, and the heart and artery beats soon become more feeble than in health.

In milch cows, almost one of the first symptoms is a diminution in the yield of milk, which becomes serous in character and small in quantity or altogether arrested. The increase of temperature and the disturbed lactation are the only symptoms noticed for from 24 to 48 hours.

The urine becomes deeper in colour, containing more solids. Urea is much increased in quantity.

The disease remains for a short time characterized by these vague symptoms, then its course rapidly advances, and, during the next three or four days, more or less marked symptoms appear.

The temperature, elevated at first, does not maintain the same altitude during the whole course of the disease. It soon descends either because some improvement has taken place, which is rare, or because the subject becomes very rapidly weakened. The surface temperature is irregular, the ears and extremities becoming cold. The sick animal soon falls into a state of somnolence, stupor and coma; the eyes are lustreless and half-covered by the drooping lids. The conjunctivæ are reddish-yellow in colour, either uniform or darker in patches and ecchymosed; it is always the seat of a hypersecretion, which shows itself at first by a limpid flow from the eyes, to which soon succeeds, in a progressive manner, a yellow discharge, which by degrees becomes purulent, greenish in colour, and irritates the skin over which it flows.

As the animal becomes fatigued, the erect position becomes painful, and the sick generally asume the recumbent one and remain down.

The skin is hot, congested, and dry; sometimes it is covered with sweat, more particularly in certain regions, as at the base of the ears, etc.; the epidermis exfoliates and is easily detached; the hairs are erect; the general sensibility is exaggerated. In white-skinned animals we notice the congested appearance better, as the skin assumes a brick red colour. We may observe trembling and shivering of certain groups of muscles, and as the disease progresses the animal may shake. When the disease is of some considerable duration, locomotion becomes painful and difficult.

With regard to the digestive apparatus and the function of digestion we observe numerous symptoms in connection with them.

Inappetence becomes in most cases complete, but not always; rumination is suspended; eructations of gas are of frequent occurrence. The mouth soon exhales a fœtid odour; the buccal mucous membrane is swollen, hyperæmic, and hot; the tongue becomes furred, congested and red coloured, more particularly on its borders and inferior face. Salivation is very abundant; the saliva at first clear and limpid becomes greyish, thick yellow, viscous, fœtid, flocculent and sometimes sanguinolent; it flows from the lips, carrying with it pellicles plaques, grains or

epithelial cells of a yellowish or greyish colour. The sick animal frequently moves the jaw and grinds the teeth. The muffle swells; its epithelium softens and is removed. The buccal membrane sometimes presents, in addition to its congested appearance and its yellowish red colour, which is very manifest on the gums, the sides and inferior surface of the tongue, ecchymosed patches more or less hidden by epithelium which has become thickened. The papillæ are congested. The thickening of the epithelial layer which covers the mucous membrane of the mouth is not always regular. The mucous membrane of the mouth often presents an uneven appearance with a greater or less number of saliant points, at the level of which the thickening has been more pronounced; at these points the derm of the mucous membrane has been more congested than at others. For this reason the epithelium of the mouth easily desquamates; it detaches itself in small masses from the more thickened points and is removed with the saliva. This desquamation occurs on the surface of the whole of the mucous membrane and more especially in certain spots, the labio gingival cushion, cheeks, tongue, etc., in the form of plaques or grains. The result is that the derm is denuded at a number of points, and there presents itself of a reddish-yellow colour. The reddish colour which the saliva sometimes assumes is explained by this epithelial desquamation; the derm being denuded, its capillaries congested and distended with blood, may be the seat of hæmorrhage, and blood thus becomes mixed with the saliva. The lips are almost invariably swollen, and vesicles may be sometimes seen resembling those of aphtha and formed by the collection of serous material beneath the epithelium.

In Europe these changes in the structures of the mouth are almost invariably met with. In India, on the other hand, they are by no means well marked, but they are occasionally met with in severe outbreaks of the disease. If the mouth be carefully examined, we may observe in some cases irregular patches of congestion and invariably a dark congested appearance at the posterior part, fauces and pharynx, and in some cases erosions will be met with there; but as regards the tongue and labio gingival membrane, the characteristic appearance which constitute the pathognomonic symptoms in Europe are frequently wanting.

The abdomen becomes tender, painful on pressure; the animal looks round at the flanks, as in colic; it paddles with the hind feet which are brought well under the belly; it frequently switches the tail, and in this manner indicates the presence of abdominal pain.

The rectal mucous membrane is hyperæmic, swollen, red and infiltrated.

There are rumblings of the bowels, borbyrygma, often audible at some distance, which indicate the progress and aggravation of the disease, and that the constipation which existed at first is about to give place

to diarrhœa. This diarrhœa is at first fæcal, but soon becomes more fluid, and later is mixed with mucus or muco purulent greyish material. It becomes softer and more fœted, even entirely liquid, and containing blood or truly dysenteric. Defæcation is frequent attended with some tenesmus, and the fæces rejected with some force. In a short time the animal becomes more and more feeble and can scarcely move. The anus becomes patent, dilated, and the fluid excrement escapes involuntarily, soiling the tail and hind legs.

The symptoms furnished by the digestive apparatus are most important and easily observed.

There is invariably a rattling and harsh cough present.

The respiration, at first scarcely altered, becomes gradually embarrassed, sometimes attended with a roaring sound, and the expired air at first warm soon becomes cold and fœtid. It assumes a peculiar fœtid odour, which is exhaled from the animal generally, and also from its dejecta, and which is quite characteristic of the disease.

The pituitary membrane is the thickened congested, red or spotted, and always the seat of a pronounced catarrh. The discharge is at first serous, limpid; then it becomes thicker and yellow in colour, greyish, reddish or green; it is very irritant and excoriates the part over which it flows. The circulation is more or less accelerated, according to the severity of the disease; the pulse may beat from 120 to 140; it becomes weaker as the animal gets feebler and more debilitated, and death draws near.

The blood is gravely changed; it contains the colouring matter of the bile, which explains the reddish-yellow coloration of the mucous membranes. The capillary system is much congested, which explains the hyperæmia and coloration of the integument. The skin, conjunctivæ buccal, pituitary, rectal and vaginal mucous membranes are generally congested and reddish-yellow, or marked with spots of extravasation; vesicles are often present in the mouth, vulva, and lips, which are swollen.

Lactation, which was only diminished perhaps at the commencement of the disease, soon becomes almost or completely arrested: the teates are soft and give nothing but a serous product in small quantity.

Localization of the disease in the nervous centres is by no means uncommon even in India, and when it does occur symptoms of vertigo, excitement, coma, somnolence or an access of fury, or even sometimes rabiform symptoms, are manifest.

Rinderpest may, as has been already stated, affect various form according to the different races of aminals, seasons, and *regime* of the animals it attacks; it varies in different epizooties, and a host of other conditions influence it. Hence it may assume a benign form in certain countries, in which it is always present, such as the Steppes, India, China, Thibet, Persia and the like at certain seasons of the year, and under such circumstances the skin symptoms are most prominent, there

being moderate derangement of the bowels, and it very frequently terminates in recovery. Cases having been reported in which as many as 75% of the animals attacked recovered under treatment.

It is by no means unusual to find animals in this country suffering from the disease presenting very slight and badly marked signs. The only symptoms in certain cases are simply elevation of temperature, congested skin, redness of the conjunctival mucous membrane and the pituitary, a discharge from the eyes and nose, moist rattling cough, slight dullness and diarrhœa. There may be complete absence of the so-called pathognomonic symptoms, and recovery takes place in a few days. In more severe cases in India we have the above symptoms aggravated, the diarrhœa becoming dysenteric, and an eruption on the skin resembling in some measure a variolous eruption; but such cases are also mild, and the eruption is generally considered to be a favourable symptom. There is a fairly good description of the more severe form, or the malignant one as we may term it, given by an agriculturalist in India, which is a practical, accurate one as regards this country: "In milch cows the milk dries up, and this occurs two days before the appetite is lost; after this purging sets in with a discharge from the eyes, mouth and nose of a watery character; the breathing now becomes very laborious, with an offensive odour, and the pulse much accelerated, with every indication of a highly inflammatory state of the system; the tongue and mouth are free from sores, and no derangement of the structures can be detected. The excrement, which was previously thin and watery, now becomes mixed with clotted blood and mucus; at this stage the animal loses strength and manifests signs of great suffering. The discharge of blood and mucus becomes more frequent and accompanied by severe straining, the difficulty in breathing increases, there is occasionally severe coughing; the eye loses its brightness, the animal becomes more and more restless, finally dying amidst convulsions." Ninety per cent. of this gentleman's cattle succumbed, although every effort was made by him to save them.

The following are the symptoms described by Veterinary Surgeon Thacker in 1866, as noticed in Southern India: "The first marked sign is a peculiar spasm of the muscles of the front of the chest and neck, sometimes also of the hind quarters. This is apt to be mistaken for shivering, which not unfrequently accompanies or succeeds it. Rumination ceases, and in cows the secretion of milk is stopped: the coat stares, the ears droop, the back is arched, the hind legs standing under the body. In the early stage the temperature rises to 105° F., but falls to 88° as debility becomes marked. The pulse is at first but little disturbed, but with the development of the disease it rises to 100 or more, becoming progressively smaller and weaker. Respiration is somwhat quickened, and when the respiratory muscles become involved is laboured.

"The bowels at first constipated, afterwards become relaxed, followed by a most fœted watery purging, and as the disease approaches a fatal termination, small evacuations of green mucus mixed with blood are passed. The disease proved fatal in from 24 hours to 8 days."

In an outbreak which occurred at Kurnool, Veterinary Surgeon Thacker gives the following description, which is simple:—

"*First stage.*—The attack generally comes on gradually, evidenced by occasional shivering fit; the appetite less; animal appears dull, with drooping ears and a rough staring coat; the bowels costive; rumination ceased or slightly performed, eyes weeping, pulse quickened.

"*Second stage.*—Appetite gone; nose dry and hot, and commencing to discharge thick mucus and a discharge of mucus from the eyes; purging commenced; lining membrane of the eyelids of a dark red colour; pulse quick and small.

"*Third Stage.*—Generally lying down from weakness; purging violent and very offensive; fæces, mixed with slimy mucus and blood, is passed frequently in small quantities and attended with straining; the eyes become sunk, the countenance anxious, general restlessness, partial insensibility, and death."

Mr. Thacker in his report to the Government of Madras notes as follows with regard to a disease called "Kudimow," which differs from malignant Rinderpest:—

It is essentially a disease of a low febrile character, infectious in the highest degree, insidious in its attack, making a considerable progress before any untoward signs lead to apprehension on the part of ordinary observers. The appetite continues but little affected for the first 3 or 4 days; a dullness about the animal, a slightly heightened temperature of the body perceptible by the thermometer, and a somewhat rougher appearance of the coat being the premonitory symptoms. These are followed by shivering fits in the colder parts of the day, but they are unattended by the peculiar twitching of the muscles of the neck and flank observed in malignant Rinderpest. The further progress of the disease is marked by entire loss of appetite, and increase of temperature over the body now apparent to an ordinary observer, drooping ears, coldness of the horns, ears, and legs, evacuations indicative of defective digestion, pulse full, ranging from 60 to 80, and in occasional cases to 90, slight congestion of the lining membranes of the eyelids, and rumination only occasionally and imperfectly carried on. The last stage is ushered in with diarrhœa, the evacuations being of a very watery character, and in occasional instances mixed with blood. The pulse loses power, being almost imperceptible excepting at the heart, but continues unaltered in number; respirations are quickened, temperature falls

below the normal; patient becomes restless, weak, lying down; the eye sunken, the countenance anxious, the senses somewhat aberrant; 70 per cent. succumbed. The above is an excellent description of the form assumed in very many outbreaks.

In other countries the disease is always severe and more fatal at its first onset, and presents during its course two successive periods—a congestive and a typhoid period. The first is characterized by an elevation of temperature, acceleration of the pulse, and congestion of the integument; then the second period comes on, and we observe coma, somnolence, stupor, weakness, and rapid emaciation. The line of demarcation between these two is always difficult to determine.

At the commencement of the malady there is always prostration or excitement. The integument and mucous membranes may not present the icteric tint in all cases; sometimes their coloration is not uniform, but they may be studded with yellow, red, or black patches.

The discharge from the nose sometimes becomes sanguinolent, and epistaxis may occur. Pulmonary emphysema is not uncommon, nor is subcutaneous emphysema. In rare cases pulmonary congestion and emphysema may lead to the confounding of Renderpest with lung and chest affections. Simple emphysema of the lung, however, is not attended with congestion of the integument, sanguinolent nasal discharge, etc. By auscultation and percussion it will be clearly distinguished from peripneumonia. According as the various symptoms predominate, we say that Rinderpest affects nervous, vertiginous, comatose, furious, bronchial, pulmonary, gastric, or enetric forms, but as a rule, we meet with several of these forms combined.

In some epizooties we observe the cutaneous form more prevalent, and especially in parts of India; we then get a variolous exanthematous, vesicular, pustular or phlegmonous eruption on the skin.

Renderpest may on some occasions be complicated with foot and mouth disease or with anthrax; it may co-exist with pneumonia or tuberculosis, etc., and in such cases it is exceedingly grave.

In females at an advanced stage of gestation it nearly always provokes abortion.

The course of the disease varies according to the form it affects; it is generally rapid, in severe cases terminating in death after the lapse of 4, 6, 8, or 10 days. Animals become rapidly debilitated in this disease and they fall into a state of somnolence and stupor, which is very marked; nutrition is arrested; emaciation is very rapid; the sick are surrounded by a fœtid atmosphere. In the last stage of the disease the saliva, discharge from the eyes and nose thicken and become sanguinolent; the diarrhœa becomes seromucous, sanguinolent and very fœtid. Finally, the individuals from want of strength remain constantly in the recumbent position, the head resting on the ground; the respirations

become deep, and the animal after short agony succumbs amidst convulsions.

Rinderpest does not always terminate fatally. In India and Russia and in other eastern countries very large numbers sometimes recover from the malady, but the mortality seldom is less than 50 per cent., unless in extraordinarily slight cases. When recovery takes place after a serious attack, which is rare, it is always preceded by a long period of convalescene, during which relapses may occur, and after which certain permanent lesions may remain. The benign form of the disease is most frequently followed by recovery: animals apparently recovered must be considered as suspects until fifteen days have elapsed since recovery, because virulent matter may remain on the surface of the body for this time. When an animal begins to progress towards recovery, the first sign indicative of improvement is an increased quantity of fæculent matter in the evacuations, which gradually assume a more consistent character; the pulse becomes softer and fuller, then the appetite begins to return, the warmth of skin natural, and lastly rumination and the secretion of milk returns.

Rinderpest resulting from inoculation differs but little from the spontaneous disease; the symptoms are the same; its medium period of incubation is from 5 to 6 days and may vary between 3 and 21 days, and its severity is about the same. The virus of Rinderpest does not appear to be attenuated by successive cultivations, and inoculations during an epizooty cannot be recommmended at present, although some cases of the inoculated disease are of the benign form.

Rinderpest always assumes an epizootic form in a country into which it is introduced; it is rapidly transmitted from the first sick to other animals and spreads from the first stable to others, over localities and districts, etc., tending always to propagate itself and not to disappear.

The disease varies according to season, in India being especially noticed in some parts at the end of the hot weather or after the first rains; it extends more easily in moderately warm weather and is more fatal in winter; excessive heat seems to modify its severity and limits its spread. In places where it is enzootic, it is not generally fatal.

Symptoms of inoculated Rinderpest (Fleming).—The symptoms of the inoculated disease as witnessed in the Steppes are said to differ from those which appear when it has been produced in a natural manner. In the former, the first manifestations are witnessed after a variable period, from the 3rd to the 6th day, very rarely beyond the 8th and 9th. The earliest indications are fits of restlessness, tossing the head, whisking the tail, pawing and kicking, and, at intervals, a slight dry cough. Then succeed dulness and prostration and a desire to separate from the other animals; the beast lies extended at full length, general

and partial tremblings are observed, and shiverings, particularly in the ulnar and patellar muscles; a paroxysm of fever sets in, which lasts from one to six hours. After the paroxysm, which at times terminates the disease, the animal gets up and its liveliness and appetite return; but there may occur a relapse of greater severity, and when these relapses persist uninterruptedly, it is an indication that the fever is assuming a putrid character. The pulse becomes small, quick, irregular and intermittent, and is imperceptible towards the close of the malady. The region of the loins is very sensible, and the animal nearly falls when pressure is made at this point; the visible mucous membranes are dry, at first red coloured, and, if the disease continues, subsequently of a pale yellow, then a bluish hue; the matter discharged is thick and stringy, the eyes are tearful, the muffle hot and dry, the ears, base of the horns and limbs are cold. To constipation succeeds a serous diarrhœa, which soon becomes frothy, fœtid and blood-tinged, and is alternated with tenesmus. The respiration is difficult, plaintive, and at last the animal lies or falls down, throws the head round to the side, and stretching out its limbs rigidly dies, most frequently without any convulsive movement. Cows in calf nearly always abort before death occurs. When emphysema appears about the back, it is nearly always a very unfavourable sign; according to Stephannoff, the inoculated disease presents this peculiarity—that it is nearly always fatal when the symptoms appear four or five days after inoculation. Before the 15th day the crisis occurs; if the animal lives beyond this period, recovery is certain in the majority of cases. The progress of the malady according to the same authority quoted by Reynal, is influenced by age, sex, the physiological condition of the animals, their individuality, the season, temperature, and state of the atmosphere. Therefore it is that young animals withstand its effects better than adult and bulls and cows in calf. Those seasons which are uniform in temperature, as the summer or winter, are more favourable for inoculation experiments than spring or autumn. The operation also succeeds better with Steppe than other cattle, and probably this may be the case with Indian animals. In other animals, however, it usually produces quite as serious a form of the disease as that which is induced naturally.

Prognosis.—In European countries it is a very fatal disease, exceptionally so in the majority of cases. In India and Russia this mortality is much less, but still very considerable, and the losses which result are considerable on account of its nature and the insidiousness of its attack. The prognosis must necessarily be very guarded, and will depend on the severity of the attack, the locality, and the form in which the disease manifests itself. It is more hopeful in the plains after very hot weather and less so in the hills in moderately warm weather. It is also more likely to terminate in recovery when it appears in the form which is marked by considerable skin eruption, etc.

Diagnosis.—When the disease exists in a district and we observe its introduction into a village, its diagnosis is not a very difficult matter, as we have to guide us what can be designated logical deduction in addition to the symtoms manifested; but generally, in the absence of precise indications, the first cases are not easy to detect, as the symptoms at first are not diagnostic. If, however, there is any reason to suspect the existence of Rinderpest, either from any indication of its presence or from presumption arising from the probable existence of anterior cases, or of the importation known or suspected of animals sick or suspected, we should always exercise prudence and take such precautions as to secure against its probable existence, for we shall not for long be kept in doubt as to the certainty of the existence or otherwise of the disease.

We should in all suspicious cases seek to obtain all the information possible as to whether Rinderpest exists in neighbouring countries or districts, whether the animals have been recently imported, etc., etc. We should never neglect to carry out *post mortem* examinations on any animals which may have died, for the lesions of typhus are very important and may contribute much to establish the diagnosis. If information on these various points is not forthcoming, we must direct our attention to the sick animals themselves, taking the necessary precautions, such as sequestration, isolation, disinfection, etc., to prevent extension of the malady; but the diagnosis becomes easy when the symptoms, vague at the commencement, become accompanied by shiverings, trembling, congestion of the integument, flow of tears, discharge from the nose, desquamation of the buccal membrane, diarrhœa, stupor, and other symptoms which characterize typhus. We may then be almost certain as to the nature of the malady we are dealing with, and in order to remove any doubts we may have on the subject and to confirm our diagnosis, we may examine the lesions at *post mortem* examination or, if necessary, have recourse to inoculation.

Lesions.—The lesions of typhus are very variable as to their number, seat, extent, intensity, according as to whether the malady has been fully developed or is in a more or less advanced stage.

The principal lesions have their seat on the mucous membranes of the digestive passages, of the respiratory organs and of the urinary apparatus; we may also meet with them on the skin, in the subcutaneous connective tissue, muscles, nerve centres and circulatory apparatus.

The lesions of Rinderpest always present certain general characteristics; they show themselves more particularly in a red, reddish-yellow, brownish or blackish colour, uniform and regular, or marbled or irregular and studded over with darker coloured spots. This coloration is due to hyperæmia of the tissues and changes in the blood, and it is accompanied by a more or less abundant exudation, bringing with it a certain quantity of the colouring matter and soaking the tissues. When the coloration is

not uniform, when it is marbled, there are points where stasis has occurred with rupture of the capillaries and hæmorrhages. The congested state of the integument and of the various organs which explains the coloration already mentioned is itself produced by the action of the blood, which becoming altered is irritant, over excites and paralyses the vasomotor nerves, from which results a dilatation of the capillary vessels and congestion, which is followed by an exosmotic movement leading to effusion of the blood plasma and numerous cellular elements.

The hyperæmia which takes place on the mucous membranes is always followed by hypersecretion, which shows itself by the production of the morbid matter constituting a sero-sanguinolent or muco-sanguinolent discharge. The catarrhal state is accompanied by a very active epithelial desquamation; this is a constant characteristic; it may be regularly distributed over the surface of the body, or else it is more profound at certain points.

Vascular ruptures are produced after the congestion; and besides these ruptures an infiltration due to exosmosis of the blood plasma always forms in the sub-mucous connective tissue. This infiltration is more or less pronounced and is very manifest in certain organs (abomasum and small intestine), and it explains the turgescence which the mucous membranes present.

The exosmotic movement gives rise not only to the passage of cellular elements to the surface of these mucous membranes, but it also establishes an inflammatory condition; the cellular elements proliferate, and the cellules so formed at the surface of the inflamed part undergo rapid granulo fatty degeneration or are transformed into pus elements.

The condition of the cadaver is variable, according as the animal has died a natural death or has been slaughtered before the disease has run its course. During the course of Rinderpest emaciation rapidly comes on, commencing at the hind quarters.

The eyes are bleared, the nasal discharge purulent, saliva fœtid and thick, the diarrhœa flux more or less abundant. After death we note more or less emaciation; the natural orifices are soiled with morbid products which flow from the living animals; the cadaver exhales a characteristic odour of a very fœtid nature. The eyes are more or less dark coloured and are bathed in a mass of purulent matter. Eruptions are visible on the skin, besides pustules, phegmonous tumors, etc.

Subcutaneous œdema exists, more abundant at the depending parts. In the subcutaneous connective tissue we find gas when the disease had present symptoms of pulmonary and subcutaneous emphysema. The inner face of the skin is always more or less reddish-yellow in colour, and black and incoagulable blood flows from the cut vessels when the disease is advanced. The subcutaneous connective tissues are congested reddish-yellow in colour, presenting dark patches of brown or black

ecchymosis; these changes also exist in the interstitial connective tissue of muscles in the perinervous and perivascular tissues. The meat will then present special characters when the animal has succumbed to the malady. The articular synovial membranes and the tendinous ones have a wine-coloured, brownish, uniform or spotted hue, and contain a sanguinolent serosity.

The muscles are sometimes little or not at all altered, but in those animals which have died of the disease they are profoundly changed, softened, their fibres altered, some having undergone colloid or granular degeneration. Certain oviform bodies which are regarded as the ova of entozoa are sometimes met with in the muscular fibres, but their real nature has not been yet satisfactorily decided. In the substance of the muscle we may meet with ecchymoses, red-yellow colour, brown or black patches, more particularly in the muscular interstices. None of the above diverse changes have any real pathognomonic value. The true lesions are those met with in the digestive apparatus; these are numerous, occurring from the mouth of the anus or throughout the whole of the digestive tract. We find a very intense state of congestion and an uniform or patchy coloration which is particularly well marked in the abomasum, in the neighbourhood of the pylorus, and in the small intestine. Everywhere the infiltration is very abundant. The catarrhal and the inflammatory state evince themselves by a hypersecretion of muco-purulent matter, accompanied by swelling and desquamation of epithelium, so that the granular degeneration of the cellular elements is brought about by the exosmotic movements or results from the cellular proliferation. In the mouth we find very characteristic lesions in Europe. The lips are always swollen more or less; their connective tissue is infiltrated. The tongue is hypertrophied; the buccal membrane is swollen. The mouth exhales a fœtid odour and contains a thick, viscid, very sticky muco purulent, fœtid saliva, rich in degenerated epithelial cellules, in embryonic elements and pus cells. The epithelium is swollen, thickened and softened; it is easily detached, and is desquamated more or less profoundly in certain more or less numerous places; it detaches itself in some regions such as the pad, lips, internal surface of the cheeks, inferior face of the tongue, etc., under the form of pellicles or plaques or of yellow or grey grains, a number of which are present in the saliva. The points from which the epithelium has been detached are the seat of intense congestion, from which results an exudation which raises the epidermis and causes it to become detached. In fact the points at which congestion has left the derm bare look congested, brownish or black in colour, and sometimes, but rarely, the points are transformed into ulcerated wounds. The derm of the mucous membrane is first changed, even before the epithelium becomes so; it is much congested, and if the red

coloration is not easy to see, this is due to the thickness of its covering At points where the epithelium is thinner the derm appears reddish-yellow in colour, brownish-black or marbled. There is always swelling and infiltration of the mucous membranes and connective tissue. The follicles and glandules of the mucous membranes are hypertrophied and congested; their secretion is more abundant and the follicles may open out and become ulcers.

These various lesions also appear in the pharynx, the epithelium of which is swollen and easily detached. The pharyngeal mucous membrane is congested and the seat of catarrh; it is covered with a purulent mucus; the sub-mucous tissue is thickened and infiltrated, and the glandules are hypertrophied. These alterations are sometimes met with, but to a less degree, even in the œsophagus, rumen, reticulum, and omasum. They are particularly frequently encountered in the rumen. The epithelium is thickened and becomes detached; the mucous membrane is uniformly congested or presents ecchymoses, spots, aborizations, intramucous hæmorrhages or sub-mucous extravasations; the submucous connective tissue is infiltrated and thick.

The peritoneum is hyperæmic, reddish, sometimes spotted, and contains a reddish-yellow serosity. In the abomasum the lesions are very varied and numerous; they contain a mucous material viscous greyish, yellowish or reddish, fœtid and very rich in cellules of epithelium, in embryonic cellules and in purulent cellules; when the product is yellowish-red the congestion is very intense. The gastric mucous membrane is the seat of a very pronounced catarrhal state which exists at the surface of the membrane and in its glandules. The thickened epithelium swollen and softened, desquamates easily, is detached *en-masse* and leaves veritable plaques uncovered, which are sometimes transformed into ulcerated wounds. These denudations of epithelium are most marked at the summits of the folds of the mucous membranes and in the neighbourhood of the pylorus. The derm is evidently congested; it is red, reddish-yellow, or brownish in colour. The coloration is irregular and has often a marbled appearance, that is to say, that spots or plaques more deeply coloured and more or less numerous are observed on the surface of the mucous membrane. Hæmorrhages sometimes occur under the epithelium and raise it; they may also occur in the woof of the submucous tissue.

These various lesions (spots, hæmorrhages, etc.) are principally seen in the vicinity of the pylorus and on the folds of the mucous membrane. We frequently meet with sphacelated plaques in the pylorus, or congestion of the mucous membrane leading to superficial or deep mortification of that membrane in the neighourhood of the pylorus. These plaques are more or less extensive and ordinarily few in number; they are dark coloured, and are sometimes already in course of elimination and

surrounded by a line of demarcation. At other times separation is complete and a wound or ulcer remains, the tissue of which is actively congested. The summits of the folds of the mucous membrane sometimes present surperficial ulcerations. The gastric mucous membrane often has a cribbled aspect from the changes in the glandules which are inflamed, congested, more salient, and have their orifices enlarged.

The sub-mucous tissue is much infiltrated. In the small intestine we find a muco purulent or caseous, greyish-yellow, or red material, exhaling a fœtid odour and very rich in elements. This material covers the whole of the intestinal mucous membrane; at the level of the solitary glands it is thickened, more caseous and adherent. The mucous membrane and its glandular apparel are manifestly in a state of catarrhal inflammation. The epithelium thickened, swollen and softened, easily desquamates and is detached in small patches. The derm is vividly congested; it has a reddish tint or is brownish or black; its coloration is irregular, and spots and points of a darker colour show themselves, especially at the summit of the folds of the mucous membrane; we observe at its surface and in its thickness more or less extensive points or patches of hæmorrhage; an abundant exudation, more or less extensive points or patches of hæmorrhage; an abundant exudation exists in all points, particularly at the follicles and Peyer's patches; the sub-mucous tissue is infiltrated; the mucous membrane thickened.

We sometimes find mortified patches similar to those met with in the abomasum, and these plaques are either adherent or in course of elimination; at other times they have given rise to ulcers with considerable loss of substance. The alteration of the solitary or agminated glands may also give rise to ulcers. The intestinal glands are hypertrophied, but what is most remarkable in the lesions of the small intestines is assuredly the alteration in the solitary follicles and Peyer's glands. The closed follicles are the seat of a very pronounced inflammation, the characters of which vary according to the period of the disease. They are at first hypertrophied, more salient and surrounded by a zone of hyperæmia; they are the seat of a very intense fluxionary movement and an exaggerated proliferation; they contain a large quantity of cellular elements which are always undergoing granulo fatty degeneration. They thus form so many small centres or caseous nodules, which soon open to evacuate their contents into the intestine; we also often observe besides, as we have already noticed, ulcers which have been formed in this fashion.

In a second phase the epithelium is ruptured and the follicles so opened evacuate, more or less completely, their caseous contents into the intestine. Sometimes the product, caseous and yellow, still adheres in larger or smaller quantity to the surface which has secreted it, and we then observe the presence of a yellowish matter at the centre of the cupuliform wound which is the result of ulceration of the follicle. At

other times the product has been completely removed, and the ulcer remains clearly cupuliform. In the third period, then, the follicles are transformed into characteristic ulcerations, the borders of which are salient and somewhat indurated; they are always encircled by a reddish areola, which may, however, be brownish or black.

The hyperæmic zone which encircles the follicles is reddish at the commencement, but becomes later brown or black in colour, and is seen in this state when the disease has been of some days' duration. Peyer's patches are hypertrophied, always covered with a greyish viscous material which is produced in abundance and may completely hide them. They are surrounded by an areola which is first red, then brown or black. The elements which compose them present the same alterations as the analogous changes in the mucous membrane. The follicles are hypertrophied and hyperæmic, and give to the patch the aspect of a fragment of skin; they are turgid, sometimes ulcerated, and may present the same characters as those which are disseminated on the surface of the mucous membranes. The villosites are hyperæmic and their epithelium is often desquamated. Brown pigmentation, or even black, is seen in the solitary glands around the agminated patches and the ulcerations; we see it also at other points of the mucous membrane under the form of spots, plaques, etc. This pigmentation by degrees replaces the red coloration of the commencement; it shows itself when the disease has lasted for four or five days, and is consequent on modifications which occur in the colouring matter of the blood.

In other parts of the intestine (cæcum colon, rectum) we observe alterations much resembling those of the mucous membrane of the small intestine. We also find a muco-purulent, viscous, very adherent greyish-yellow or reddish material rich in cellular elements.

The epithelium is swollen, softened, and in course of desquamation; the mucous membrane is hyperæmic and the sub-mucous tissue is infiltrated. In the cæcum we sometimes meet with a caseous material of a fibrinous appearance exuded and adherent to the surface of the mucous membrane at several points. This material presents itself under the form of filaments, and if we detatch it a denuded point remains which is greatly congested; it appears to be produced by an exudation which is established from certain ecchymosed plaques.

The liver may present certain alterations, being sometimes yellow or earthy yellow in colour and its cells undergoing degeneration.

The mesenteric glands are hyperæmic and congested; they are red or brownish coloured and softened.

The mucous membrane of the respiratory passage is more and more altered.

The pituitary is hyperæmic, reddish yellow; its tint is uniform or marbled with darker spots, points or hæmorrhagic plaques; it is

catarrhal and covered with a yellowish muco-purulent and greenish coloured product; it is thickened and its sub-mucous tissue is infiltrated; its epithelium is desquamated, and it sometimes presents wounds with loss of substance and covered with a pseudo membranous product.

On the laryngeal mucous membrane as also the tracheal and bronchial, we find the same alterations (hyperæmia, uniform or marbled coloration, catarrhal condition, muco purulent products, etc.)

The lung is more or less hyperæmic, and its interlobular connective tissue is the seat of a more or less abundant exudation. There is pulmonary œdema and sometimes more or less extensive interlobular emphysema. The bronchial ganglia are tumefied, hymeræmic, reddened and softened.

The pleuræ have a reddish icteric tint either uniform or spotted; they contain red coloured serosity.

The kidneys are sometimes tumefied, hyperæmic, softened and friable. The vesical mucous membrane is injected and turgid. The utero vaginal mucous membrane is congested, infiltrated, thickened uniformly red as spotted and catarrhal. The mammary glands are flabby and infiltrated.

The brain, spinal cord and nerves present more or less vivid hyperæmia and an exudation into their connective tissue.

The lesions of the circulatory apparatus are little pronounced at the commencement. The blood, at first but little changed, undergoes important changes during the course of the malady. It becomes black and loses its coagulability at the end.

The white corpuscles increase in number and "b onnets" are present in it, the signification of which is at present not well understood. The pericardium is congested, reddish coloured and spotted; it contains sanguinolent serosity. The heart is yellow, pale coloured and softened; the endocardium offers ecchymosed patches, which also exist in the substance of the heart; the interior of the cardiac cavity and of the vessels is red and icteric. In all organs the capillary network is relaxed, turgid, and hyperæmic. The pathogeny of all the lesions is explained by the alteration of the blood. The altered circulatory fluid acts on the elements and on the vasculo motor nerves paralysing them; the vessels then dilate and blood stasis occurs which results in the exudation, infiltration, and dissolution of the coloured cells. At the surface of the integument where exudation is active we observe a catarrhal state accompanied by a ploriferation of the cellular elements and their rapid degeneration.

Etiology contagion.—Rinderpest is a malady which always develops by contagion; this is pretty well established at the present day; it is absolutely false to say that it may develop spontaneously. In Eastern countries like India it exists as an enzootic and perpetuates

itself by contagion. Reynault thus gives a *résumé* of his conclusions: "Rinderpest propagates itself exclusively by the very active contagious properties which affected animals; the products of their excretions and their debris when dead possess properties which impregnate other animals, people, and things which have touched or approached them, their excretions or their debris, and which these may transport to other animals. It is only when they have been exposed to contagion, when they have been brought into contact in some way with one or several of the impregnated agents, that our animals can contract Rinderprest. But these agents are humerous when sick animals and healthy are living in the same fields or only in neighbouring fields, in the same stables or in adjacent ones, frequenting roads, or drinking the same water, mixing one with another on the highways or at fairs or markets, etc. They are always dangerous, but at the same time easy to avoid, when sick and healthy animals inhabit fields of farms or villages a little removed from each other, if all communication whatsoever is not completely suspended between the animals, the people and things which after touching or approaching the animals and places affected, approach and touch shortly afterwards healthy animals." Contagion being recognized as the sole cause of Rinderprest, we must study with care the nature and characters of the contagion and endeavour to determine the mode and conditions of its transmission. Cantagion, as we have said, is the cause of Rinderpest, as has been clearly proved by numerous observations and from the fact that we can transmit it from one animal to another.

It is permanently located in certain portions of India and the steppes of Russia, and in these countries it is relatively benign, so much so in fact that the animals attacked sometimes appear little sick or not at all, and in this way it often happens that a dangerous animal, or one suffering from the disease, may be transported to a country or place and disseminate the disease amongst healthy animals. This very frequently happens in India, especially in those parts of the country where it is the custom to drive away the apparently healthy animals from an infected village to other grazing grounds where they may come in contact with other healthy animals, and for this reason it is that this mode of proceeding has to be condemned. From the above facts it will be evident how very easy it is for the disease to be spread from place to place.

Contagium.—The contagium of Rinderpest exists in the whole organism, in all the solids and liquids, in the fluids and products of normal secretions, in the lymph, blood, saliva, urine, milk, tears, etc. It is abundant in the products of pathological secretions, in the discharge from the nose, tears, in dysenteric and diarrhœic materials, etc. All the products of a diseased animal are virulent; also the products of the bones, muscles, organs, normal fluids, blood, lymph, milk, serosity,

morbid products, etc., contain the contagium and may propagate the disease.

The nature of the Rinderpest virus seems to have been determined according to the later researches in Russia, and it appears to be believed that it is a parasite of the bacterian order.

Semmer in 1871-74 observed the presence of a micrococcus in the organs of animals attacked with Rinderpest. The same observation was made by Klebs in 1872, who considered that a micrococcus was the active agent in the production of the disease. This micrococcus, which is found in the saliva, and is fixed in the epithelium determining its softening, gives rise to salivation, extends in the glands and passes into the vessels, where it forms heaps and provokes stasis, hæmorrhages, etc.

Semmer, like Klebs, had found micrococci in the blood, tissues, secretions and excretions, and had considered them as the contagious elements of the disease. Placed in contact with the mucous membranes, these micrococci penetrate into the blood, multiply, are arrested in the capillaries, determine stasis, extravasations, hæmorrhages, spread in the tissues and lead to changes. Semmer and Archangelski have established the *role* which these micrococci play in the production of the disease. Having experimentally transmitted cattle plague to the sheep, they have found micrococci in the ganglia of the animal after death and have sown them in sterilized broth and cultivated them in contact with filtered air at a temperature of 37° C. A cultivation at the third generation inoculated into a sheep gave fever, after which the animal resisted the effect of the inoculation of a second and a first generation heated for an hour to 46°—47°C. A second sheep inoculated at the commencement with a cultivation at the second generation had simple elevation of temperature, and after recovery resisted virus of the first cultivation. Five months afterwards these two sheep were inoculated with fresh virus at the same time as a calf and a fresh sheep: the two inoculated sheep were not sick; the new sheep became sick, but recovered; the calf died of Rinderpest. The virus taken from the dead calf was inoculated into the three sheep but caused no disturbance; since then however a calf inoculated with cultivated virus died of the disease.

The conclusions which may be drawn from these experiments are—that the contagium of Rinderpest is a micrococcus, which can be cultivated and attenuated by cultivation at a certain temperature and in the presence of filtered air. It is killed by—15° to—20° C and by 55° C. (131°F.).

Kolesnikou has found micrococci and bacilli, also recurved "batonnets," in the blood; bacteria and long filaments furnished with spores in the mucous membrance of the stomach, intestines, mouth and in other organs.

Kostitchschew has found micrococci and bacilli. Saulljew by cultivating the blood of the heart of an animal dead of Rinderpest in sterilized bouillon has obtained micrococci and bacteria inoculable to mice and rabbits. He considers that the pathogenic microbe of Rinderpest is a bacillus, which is transformed into a micrococcus and again assumes the bacillary form under certain circumstances. It gives a greyish-white colour on Agar Agar, becoming afterwards citron coloured, then red. The greyish-white colonies are the most virulent.

M. Piot, Veterinary Surgeon in the Egyptian Administration, in 1883, announced that he had succeeded in attenuating the virus of Rinderpest by heat to such a point as to give an inoculated animal only a slight attack of the disease, after which, however, it became immune or protected.

Whatever may be the intimate nature of the virus of Rinderpest and of its attenuation by cultivation and by heat, we are certain that it exists in every part of the animal body, in all the organs and tissues, in all the fluids and in all the secreted and excreted matters—a fact which proves that it is produced in enormous quantity by the sick animal, and that one sick animal may in this way furnish an enormous amount of it and thus form a very powerful centre of infection. The virus is rejected from the animal continuously either by way of the skin, nasal, buccal, genito urinary or respiratory apparatus, etc., etc. If we add that it may be suspended in the atmosphere either by adhering to particles of floating matter or suspended as dust, we may conceive that an animal sick of the disease is surrounded by an atmosphere contaminated and contaminating. The major part of the rejected virus contaminates the litter, mangers, food, water, etc., etc., and under these circumstances may, under favourable conditions, be preserved for a very considerable period of time. A sick animal is therefore dangerous not only itself but also on account of the solid, liquid, and gaseous materials which it contaminates.

When the virus of Rinderpest enters the system, it soon makes its action felt, multiplying and giving rise to the first symptoms. The medium period of incubation is from 4 to 10 days, but a longer period from 20 to 30 days has been noticed (Lemaitre). The virus multiplies and causes changes in the blood, which are soon accompanied by other changes, chiefly localized in the integuments.

The question has often arisen whether the animal is dangerous during the period of incubation, and at what date the virulence appears. The answer to this question seems to be that very shortly after the introduction of the virus into the system the virulence appears, so at least the fact that a bull transmitted the disease during the period of incubation would tend to show.

The experiments made by Semmer, and the post mortems of animals inoculated for only 36 hours and which have evinced no sign of disease,

have shown that lesions quite characteristic of the disease were present. This is very significant and would naturally lead us to the conclusion that an animal is virulent and capable of transmitting the disease before any symptom has been noted—a most serious matter for our consideration in carrying out measures for suppression.

The virulence persists during the whole course of the disease, and does not disappear even after death. It has been stated that it becomes attenuated as death approaches, but there is great difference of opinion on this point. However this may be, one thing is certain, and that is, that it persists for a certain time after death.

What are we to say regarding animals which are recovered? In this regard we may remark that in Europe few animals recover at the commencement of a serious outbreak, and the same is the case in India in serious outbreaks, although when the disease is of less intensity large numbers do recover. The disease here, especially in the hot arid plains where the temperature of the atmosphere rises to a point which is acknowledged to distroy the virus, or at any rate to greatly attenuate it, very frequently assumes a mild type, a benign form in fact, and large numbers recover. This is equally the case where the disease has approached the termination of an outbreak and the virus has apparently become naturally attenuated. In such cases are we to consider that virulence ceases when cure is brought about or even after this period? To reply to this question is not an easy matter. We may, however, consider that virulence persists until the disappearance of the diarrhœa, discharge from the eyes and nose, and salivation have entirely ceased; but we are not in a position to say whether the virulence persists after complete recovery; in fact there is reason to believe that it does not. Whatever the case may be, we must consider convalescent or recovered animals as dangerous for a fortnight at least, as the virus may be borne on the surface of the body in a virulent form for that period, and of this fact numerous examples have come under my own notice.

The Rinderpest virus rejected by the diseased animals, as well as that of the cadavers, may be conserved for a considerable time; its vitality seems to persist more or less according to various conditions, but however bad the conditions may be it may live for several days. The study of this question is a very important one, as it dominates the application of certain sanitary measures. How long does the virus live? On this point the observations vary very considerably, are numerous and contradictory. According to some observers the virus is preserved for days, months, and even years; whilst in the opinion of others whatever the conditions, the preservation cannot exceed 30 days; and further this cannot occur unless the virus is preserved in sealed tubes. It has been pretended that in buried carcasses the virus may be preserved for months (Victor D' Azyr), or even during years, and that

it is consequently dangerous to let animals graze where the cadavers have been interred. It has also been stated that the cadavers of Rinderpest animals exhumed after some months of burial, may still transmit typhus. This expression of opinion does not seem to be exactly true; it is perhaps much too alarmist. In 1874 M. Visear D'Arras exhumed some Rinderpest cadavers after 10 or 12 months of burial, and caused ruminants to graze over and smell the debris with no result. M. Reynal did not succeed in transmitting the disease by inoculating with material from an animal 58 days after death. It appears, therefore, that the cadavers, even when buried, do not conserve the virus for such a long period as has been stated, but as the truth is not yet absolutely established on this point, it is necessary for us to act with great prudence with regard to it and to take every precaution. The flesh, fresh skins, and in a general way all the fresh debris are dangerous, and for a certain period may serve as a means of conveyance of the virus. But on this subject, as well as on that of buried cadavers, the duration of the conservation of the virus has been greatly exaggerated. The researches mentioned by Reynal give us a fairly exact idea as to the danger resulting from the skin, horns, bones, and hair of Rinderpest animals. The skin of an animal dead of Rinderpest has not any contagious property when tanned or dried. No record of the introduction of the disease into a country by the medium of tanned or dried skins has ever been published, and hence it is not necessary to prohibit the importation of dried or tanned skins from a country where Rinderpest exists, provided it is at some considerable distance. In 1745, Courtioron tried to transmit Rinderpest to two cows by means of very fresh skins removed from animals dead of the disease, but did not succeed, and he concludes his remarks on the subject of the experiment by saying "not regarding the experiment which I have made as a reason to doubt the popular opinion, it may perhaps be expedient to order that skins may be kept in an infected village in a particular place and may be rendered saleable after a certain period has elapsed." In 1769, Camper in Holland placed the fresh skin of a Rinderpest cadaver in close proximity to two calves, but did not succeed in transmitting the disease to them by this means.

Victor D'Azyr made attempts at transmission but was not successful: he, however, did not consider his experiment conclusive, and counselled the burial of animals dead of the plague in their skins. Other experiments made by Camper in 1769 by inoculating with fragments of fresh skin proved that six days after death of the animal they may retain their vitality. Spinola, from what he has himself seen and from what Russian authorities state, believes that fresh skins coming from a neighbouring country may be dangerous, but not those from a distant country. Buniva has observed the propagation of the disease by means of the hides. It has for a long time been known that fresh skins are a fruitful source of the

propagation of the malady, but how long do they remain dangerous? According to an experiment made by Camper they are virulent six days after the death if the animals have been buried. According to Victor D'Azyr, scraps of skin may cause the disease six months after the animal has been buried. Loringer considers that fresh skins maintain their virulence for eight days after their removal from the cadaver; hence it follows that skins coming from a distance are not dangerous, especially if well dried and salted.

Does the Rinderpest virus preserve its virulence for any length of time on the surface of the litter, dung, forage, mangers, in the water, discharges, etc.? We are not in a position at present to offer an opinion on this point, although it is an immensely important one. It appears that the virus deposited on the surface of solid bodies cannot be preserved for more than four or five days in the summer or 15 days in the winter. And even this time may be abridged if they are exposed to a continuous current of air, and if at the same time they are submitted to the action of heat and the rays of the sun, or if the weather be cold and humid, for then the process of putrefaction rapidly destroys it. In these various cases the virulence does not extend over many days, generally about 11, at least so it appears. The virus found in a stable where not exposed to currents of air, and where the air is not changed, may remain virulent for a longer period, according to some observers 8 to 10 days. Placed in a flask it preserves its virulence for from 4 to 8 days, and if the flask be hermetically sealed it does so for a slightly longer period. It may also remain virulent for a longer time, 12 to 30 days, if hermetically sealed in a vaccine tube or placed between glass slides, particularly also if placed in vacuo; but in any case the virus remains virulent for a much shorter period than does that of sheep-pox (variola ovina) or cow-pox (variola vaccinia). The virus may preserve its virulence in water for a time, the length of which remains to be settled. In the dung, which is the richest receptacle, the germs may be rapidly destroyed by putrefaction; at the end of a month they have been found to be innocuous. Nevertheless it is necessary to take into account the assertions of those who attribute to the virus of Rinderpest a more prolonged virulence, and of these the most rational appear to be those of M. Chauveau. "The expired air," he says, "is not the excretion which is richest in virulent elements; indeed, it has been experimentally shown that it is poor in comparison with others. The tears, discharge from the nose, saliva from the mouth, milk, urine, and excrement more particularly contain an enormous amount of the virus, and unhappily the pernicious properties rejected on to the litter, dung, on the roads, stables, pastures, etc., may remain virulent in the open air for several weeks. These are the most dangerous vehicles of the virus, and those of which it is most necessary to destroy the contagious action." But what can we think of those pre-

tended cases in which cases of transmission obtained after prolonged dessication for several years? What credence is to be accredited to the opinion which holds that infected stables may remain dangerous during several years. What conclusion are we to draw from a fact of transmission attributed to the administration of an infusion of hay taken from a stable where the disease had been present five months previously? What importance are we to accord to successful experimental inoculation with material kept for a year? Is it not rational to think that there has been some exaggeration, or that coincidences may have led to error? Is it not probable that if the virus enjoys the power of so conserving its virulence, occidental countries to which the disease is imported could be only freed from its ravages with the utmost difficulty, for notwithstanding that a good system of disinfection be adopted, it is extremely difficult to attack all the germs. We have already seen that Rinderpest virus does not resist intense cold nor a temperature of moderate elevation, and hence is only preserved in the stables, during the severe weather of the more elevated and cold ranges of the Himalayas. The experiments of Semmer and Archangelski have shown that it is sterilized by cold at 15° to 20° C, and also by a temperature of 56° C. The sterilization of Rinderpest virus is hastened by putrefaction, dessication, currents of air, æration and ventilation, an elevated temperature, intense cold and light. In practice we must have recourse to these means as far as is practicable without danger. The sterilization on the contrary is retarded when the air is confined and not renovated, when it is humid and fresh, where light does not reach the virulent matter, when dessication and putrefaction are retarded.

Modes of Contagion.—We have already seen that Rinderpest owes its development and propagation exclusively to the very active contagious properties possessed by the sick, the products of their excretion and debris—properties which impregnate other animals and by them conveyed to others again, as also is the case with people and anything whatsoever in fact which has been in contact with the sick, their excretions or their debris. Animals may contract the disease after having been brought into contact in any way with an agent impregnated with the contagious element. These agents are numerous, and the danger is imminent when sick and healthy animals frequent the same places, live in the same grazing grounds, locality, village, or neighbouring ones, if all communication between the sick and healthy animal is not interdicted.

Rinderpest may be transmitted by immediate or mediate contagion and also by inhalation. The disease is transmitted by direct contact of healthy with diseased animals, and when an animal is placed near a sick one it is exposed to contamination by each of the three methods. When Rinderpest spreads from place to place, when it becomes epizootic, it is in almost all cases transmitted by mediate contagion. It passes from

one stable, farm, or village to another through the intermediary of diverse solids or liquids, food, water, etc., etc., which are soiled with contagious materials and which are smelt, licked, or ingested by animals. The malady is also, although less frequently, transmitted by the atmosphere, and in this particular it is well to decide or to enquire whether the atmosphere which surrounds the sick animal is dangerous at great distances. Accepting as demonstrated, for it is demonstrated that the air may act as a medium for transmission of Rinderpest, we should also recognize that although it has been stated that this medium is capable of carrying the *materies morbi* for a distance of 500 yards, it appears probable that it does not transmit to large distances. On this point, in the localities where Rinderpest has been prevalent, it has been noted that when due care has been taken, and the places carefully sequestrated, no cases of the malady have occurred, although in close proxmity to others where Rinderpest has claimed several victims (*Chauveau Visear*). The distance to which volatile contagion is possible would seem, therefore, to be not very great. It naturally varies according as the contagious atmosphere is very rich in virulent material, and whether the contagium bearing atmosphere has been exposed to winds or to current of air, and as to whether the malady is severe, more or less general, and whether there are any obstacles around the centre of infection. A wall, hedge, forest or hills may greatly retard the spread of the disease by the atmosphere; but I must repeat that in the majority of cases it is not the air which is the most fruitful source of spread of Rinderpest. Under ordinary circumstances the contagious zone does not exceed 20 to 40 metres. It is by ingestion that the disease generally propagates itself.

M. Chauveau says: "I have demonstrated that the spontaneous evaporation of the most virulent matter taken from a subject attacked with Rinderpest is incapable of drawing out any infections emanation. The drops of a liquid which condense on the walls of a bell glass under which this evaporation is conducted proves on inoculation to be devoid of virulence, whilst the least particle of the material from which it has been obtained gives Rinderpest when inoculated under the skin. I have found moreover, in analyzing rigorously well-observed facts, that contagion by germs sown in the air plays only a secondary *role* in the propagation of the malady to a distance. It is particularly by alimentation that this propagation is effected. The dejecta sown on the roads, in pastures, ponds of drinking water, or transported to a distance by means of animals or men who have trodden in them, infect the food and water." A very good example of the spread of the malady by the water supply is to be found at the end of this in an appendix, and came under my own observation in the Kotkhai tahsil of the Simla district in India.

It is probable that the disease in the Himalayas spreads to a great extent in this way, by the people pollutting the water-supply by throwing into the streams or their feeders the carcasses of animals dead of the malady.

It may be again repeated that it is contagion by ingestion which plays the most important part in the dissemination of Rinderpest; the virus excreted by the diseased, or arising from their cadaveric debris, being preserved by external media and introduced into the system with the food and drink; and it is easy to divine that this conditions are frequent and numerous where the disease exists in a part where sanitary and precautionary measures are neglected. Animals may infect themselves either by licking the walls, mangers, racks, and other objects soiled with the virus, or by eating food into which the virulent material has fallen, or by drinking water to which it has gained access.

The principal means, agents, vehicles, or intermediaries which may serve for the propagation of the disease are very numerous, and the disease when it is introduced into an organism nearly always gains access by means of the food, drink or air, these being contaminated in a variety of ways, serving to transport the virus—forage placed either above or below infected stables in store, grass from infected pastures, forage and litter which has directly received the virus from the sick animal, forage and litter trodden on by people who have been walking amongst the sick, plants manured with dung from infected stables, forage and litter which have directly received the virus excreted by the patient or have been in contact with the cadaveric debris.

Drinking water may also be contaminated with the virus either from sick animals drinking in it, or by the excrements gaining access to it, or by skins or flesh from diseased animals having been washed in it, or by animals being thrown into it after death from the disease, as is common in India, and more especially in the hills, or by wild animals drinking at it after a meal of the contaminated flesh.

The cadavers, skins, flesh and the various debris, so long as they are fresh, also serve to convey the disease, either by animals smelling them and thus introducing the virus into the respiratory passages, or by these products having been deposited on the herbage or forage or by being washed in water which is ultimately swallowed by animals; all objects which have served to transport Rinderpest flesh are, therefore, dangerous, being impregnated with the virus. Besides food and drinking water, various other objects may serve as vehicles to contagion when they are licked, or come into contact with animals, or when they are brought into contact with food or water. Of these, we may cite mangers, cribs, racks, all objects in soiled localities, sieves, watering places, fastenings, grooming kit, clothing, roads, fields, pastures, carriages, carts, manure and litter, urine, meat, various cadaveric debris, etc.

Carcasses badly buried which carnivorous animals may disinter, carnivorous animals after a meal on a dead Rinderpest carcass. We may easily imagine the danger which the majority of these intermediary vehicles may give rise to.

A single sick animal is capable of infecting a public drinking place whence healthy animals may become infected; similarly it suffices for sick animals to soil a pasture with their excretions and secretions in order that it may be the means of infecting other animals grazing over it five, six, or even twelve days afterwards. Pastures are rendered dangerous when they have been manured with litter which has been in contact or has been soiled with the virus from sick animals. Conveyances, railway trucks, and the various utensils which have been in contact with sick animals are dangerous, preserving their infective properties for several days. Litter fæces, etc., are very dangerous materials, being very rich in the virulent matter. Animals which smell, lick, or ingest them become infected. Animals and men who walk about on these materials carry the virus away on their shoes and spread it about as they walk. In this manner also, dogs or other animals walking about in a stable where animals are suffering from the disease get their feet, skins, and hair impregnated with the virulent material, and may afterwards soil water or forage which, when ingested by healthy animals, will induce the disease. The same is true of the human being, the owner, the veterinarian, and the men employed in looking after the sick. If these persons are not very particular to cleanse themselves, and more especially their shoes, they may, and often do, carry away the virus, and by soiling roads, forage, water, etc., become unwittingly the means of spreading the malady amongst other healthy cattle. It is really the boots of people engaged about sick animals which are the most dangerous owing to the likelihood of their cleansing being overlooked, and owing also to the material with which they become soiled bearing such large quantities of the virus; and the feet of animals are dangerous for the same reasons. Sheep may transport the virus of this diseases in their wool without becoming themselves affected with the disease; but it is probable that wool does not preserve the vitality of the virus for long, and that when it has been dried, and more particularly if also disinfected, it is not dangerous. Cadavers or debris of Rinderpest affected animals which have been badly buried constitute a danger. They may be disinterred by carnivorous animals and dragged about over the herbage and soil it with the virus, so that healthy animals grazing may ingest the poison and take the disease. Moreover carnivorous animals when devouring diseased meat impregnate their lips, hair, and paws with the virus, and may thus transport them from place to place.

Rinderpest may under certain circumstances be transmitted by direct contagion. Healthy animals may infect themselves by licking

the sick; young calves become affected by sucking a sick mother. Lemaitre has observed the infection of a bull consequent on his bulling a sick cow, although the earlier symptoms of the disease were not manifest at the time. Lastly, may not the intrauterine transmission of Rinderpest not be considered to be established by the large number of abortions which occur in sick animals? Neuhauss has observed intrauterine contagion accompanied by the development of the disease in the young animal. To recapitulate then, although contagion may be accomplished by inhalation, by accidental inoculation, and by heredity, we must look to the digestive organs as playing the principal *role* in this respect, they most frequently receiving the contagion through the medium of the food and drink. This point, which has been brought forward by numerous observers, is now admitted by all. Therefore, in epizootics of Rinderpest, it behoves us not to seek the cause of the propagation so much in atmospheric influences.

Species susceptible to Rinderpest.—This disease is not exclusively confined to ruminants. The Bovine species is undoubtedly more subject to it than others, and it is in it that we meet with the disease in the greatest intensity and with the most clearly marked manifestations, with the most decided virulence, and generally in the gravest form; but it is none the less true that the disease may be transmitted to the Ovine and Caprine species in the following order as regards degree of receptivity, *viz.*,—(1) Bovine, (2) Ovine, (3) Caprine. Cases have been observed which demonstrate the possibility of the transmission directly from the ox to the sheep and goat; and also similarly from the sheep and goat to the ox. These facts should be kept in mind, because it is rare to find only one species of animal on a farm, in a village or locality, there generally being specimens of the three cited species always present. Rinderpest is less severe in the sheep and goat; it is also less characteristic in its symptoms, which are less numerous and less pathognomonic; the percentage of deaths from this malady is far less in these animals. But sheep and goats affected with it nevertheless constitute a very dangerous element in the propagation of the malady, more especially in the Himalayas, where these animals are almost constantly on the move from higher to lower levels and *vice versâ* in search of pasturage, and it becomes necessary to prevent it in them if possible. The malady is very commonly observed in the buffalo, in which it is very fatal in India. The yak also suffers from a very severe form of the disease, as do all his crosses with the cow, and it is very fatal in them. In the year 1890 the disease was carried by the pack animals from the Kulu valley in the Punjab into Lahoul, and attacked the yaks and their progeny there in all the villages to Kyelang, leaving but one or two animals alive in its track. This was at an elevation of about 12,000 to 13,000 feet above sea level. The camel

also suffers very severely from the disease, it being very fatal in him; numerous instances of the disease in this animal have been cited by various authorities, and some have come under my own personal observation. Antelope also suffer from the disease, as do Cervidæ generally. The gooral and kakar or barking deer have been seen dying in large numbers from this disease in the Himalayas. The auroch, peccary, gazelle, and stag also contract the disease, as do also the *Bubalus arni* and the Mithun and Tsain. It has been observed in the wild pig of Sumatra, and has been transmitted experimentally to them. It may be probable that such animals as the cat and poultry even may become affected.

But it is in the Bovine species that the receptivity is greatest, although all individuals of this species do not evince it to a similar degree. It is true that certain individuals evince a greater predisposition to contract the malady and to take a more severe form of it than others do. Animals indigenous to the Steppes are no more predisposed to the disease than others; if they have it more frequently than others, it is because the epizooty is never extinct in the country; and the same is true in some districts in India, for instance, Kotkhai, Seoraj, Kulu, Kangra, Assam, etc. In the Russian Steppes, however, whether it is that the affection has lost some of its proper virulence or whether the animals evince a greater natural resistance, or one acquired as the result of natural vaccination, the disease is often found to assume a benignant form in these parts, whilst it is very grave when imported thence to occidental countries, as has been frequently observed.

The disease in some parts of India, and notably in those parts which are naturally dry, hot and arid, where the temperature of the air rises very high, is frequently noticed to be of a far milder type than is the case in other parts, notably the hills, and it has appeared probable that dessication of the virus by the great heat and dryness influences its virulence to a considerable extent, which would account for mild outbreaks with large numbers of recoveries following the reception of the virus into the system. Another point which may have some influence is that the animals live almost entirely in the open air. There can be no doubt that the receptivity of the small ruminants, sheep and goat, is very much less than that of the ox. This fact has frequently been brought forcibly to my notice in the Himalayas, where the "gaddi" shepherds keep enormous numbers of these animals, and where there are numbers also in every village. In these parts I have seen the sheep and goats kept side by side with affected animals of the Bovine species, feeding over the infected pastures and drinking the same water, but it has been only in exceptional cases that they have become diseased. The few cases of outbreaks amongst flocks of sheep and goats mixed have been slight, not spreading rapidly, considering the adverse conditions under

which the animals are kept, and causing but a small percentage of casualties. It may be therefore safely said that it is not rare for them to resist or to pay a very small tribute to contagion.

Man appears to run little if any danger either from the manipulation of the debris, or from consumnig the flesh of animals dead of Rinderpest, or from drinking their milk. It is a common practice in some parts of India, and I believe also in the other countries, for the people to eat the flesh of animals dead of the disease, and no bad consequence has ever been reported from people having done so. Dogs and soliped animals appear to be equally refractory.

Immunity and preventive inoculation.—Animals which have recovered from the disease acquire immunity from a future attack, according to some authors, for the remainder of their lives, but at least for a period of six years. I have had some instances brought to my notice of animals having a second attack after a lapse of seven years, and one instance of three working bullocks belonging to a planter in the Kullu valley having a second attack, to which they succumbed six years after the first. Other observers have found animals immune after seven years. Mr. Hallen, Inspector-General, Civil Veterinary Department, relates a case of three bullocks which had been attacked with the malady seven years previously, being stalled with animals suffering from the disease, eating their rejected forage, etc., and resisting contagion. However this may be, the consensus of opinion is that an animal once attacked and recovered does not take the disease again. The knowledge of this fact is found amongst cattle owners throughout the length and breadth of India, even in the remotest parts of the Himalayas, where the people are, as a rule, most ignorant and superstitious. An animal which has recovered from the disease in those parts where it is enzootic is worth double or treble what an unprotected one is, and the people are only too anxious to secure them.

Animals which escape death after inoculation with the virus, as it is at present occasionally imperfectly carried out, acquire immunity. Rinderpest then leaves after it a peculiar condition of the organism which renders the animals which have been the subject of it proof against a second attack for a long, though apparently somewhat variable, period of time.

These facts being within our knowledge and generally accepted, it would have been strange if some efforts had not been made to produce a mild form of the disease in animals, which would leave them immune without running any risk of the inoculation proving fatal. The discovery of such a virus would, of course, be of the utmost value in a country like India, where methods of suppression are most difficult to carry out. Armed with an effective protective virus, the lives of many thousands of animals could be saved from this courge to agriculture,

the position of the Indian farmer and cowherd be infinitely bettered, and the prosperity of the country much increased. We have for a long time sought to confer immunity against rinderpest by inoculating either with typhic virus or with cow-pox.

Inoculations with typhus virus have been made chiefly in Russia. Thousands of experiments have been made by the Government of that country, who are so deeply interested in the solution of the problem. Various virulent materials have been used, notably the tears and the nasal discharge. These materials were used sometimes in the fresh state, at others preserved for a certain time. They were diluted with water, glycerine; cultivated after the method indicated by Pessina; it has been sought to mitigate or attenuate them by passing them through the organism of the goat and sheep, but none of these attempts have succeeded, and after several years of experiments the official commission nominated terminated its operations in 1864, by saying that they were constrained to close the establishments of Orenberg and Kerson, and to abandon as useless any new attempt at inoculation, seeing that, so far, the mortality due to inoculation has been nearly as great as that from natural infection. Nevertheless, in 1870-71, when the disease decimated our cattle in France, Jacques Valserres, amongst others, sought to resuscitate the practice by publishing the ideas of a century before (1746); by Dodson at the time of the great outbreak in England, and in 1815 by Dupuy. Happily they did not find a sufficient number of proselytes.

Since these attempts were made great progress has been made in the study of contagious disease, notably as regards the attenuation of virus, and it has been sought to apply the new methods of attenuation of virus to that of rinderpest. Thus, in the summer of 1881, Semmer was sent with Professor Ranpalle to the south of Russia to apply M. Toussaint's method of attenuation. From some experiments we have made, says Semmer, it appears that by ten minutes' heating at 55° C. the contagion of rinderpest is completely destroyed. About 30 grammes of blood heated to 55° C. and nasal discharge treated in the same way were injected into the skin of healthy animals. No immunity against natural contagion resulted; four calves thus inoculated were placed amongst sick animals in a closed place and died from rinderpest. A temperature of 55° C. is therefore too elevated for attenuation (Neumann). Attempts at inoculation by Tonssaint's method were made in Egypt under the direction of M. Piot; twelve animals inoculated resisted natural contagion (Echo de Sociétés, etc., 1883) These results were encouraging, and it was hoped would bear fruit, but nothing has yet come of it. During the epizooty in England, 1865-66, Dr. Murchison assimilated variola and rinderpest as lancisi, and Ramazinni did at the beginning of last century and recommended

vaccination. But observation soon proved that the two maladies exercised no influence on each other.

It has been advanced that there was antagonism between the development of aphthous fever and rinderpest, but this has been disproved. With regard to the experiments which have already been carried out, numerous inoculations were made in Russia during the last century, as well as in the present one. The method adopted has been to take material from a case of the naturally-contracted disease, preferably from a mild one, and to pass it successively through a number of animals, with a view of weakening or attenuating its virulence, if possible, and by inoculation of the attenuated virus to produce a mild form of the disease, but one still capable of conferring immunity. This method of successive cultivation in animals has been carried on to the fifteenth generation, but it is found that the material so treated very often induces, on inoculation, as virulent and fatal an attack of the disease as the original virus. Inoculated rinderpest induced in this manner comports itself in the same fashion, and is just as severe as the naturally-contracted disease, being frequently fatal. The mortality appears to vary in different countries, but it is always considerable. In Russia it varies from 4 to 60 per cent. of the animals inoculated.

Another method which has been tried is that of selecting animals which have the disease in a very mild form and inoculating others with the tears, saliva, etc., with the idea that by so doing a mild attack of the disease would be induced; but the argument on which this method was adopted has no scientific or practical basis, and is erroneous, for, as is well known to be the case in this and other diseases (for it has already been stated that animals suffering from a mild attack may be the means of inducing the most virulent outbreak amongst other cattle), the benignity of the disease appears to depend more on individual idiosyncracy, previously-acquired immunity, or some temporary attenuation of virus which recuperates itself when passed through a susceptible animal. A good example of this is to be found in the fact that attenuated anthrax regains its pristine virulence when passed through suitable subjects. Supposing, then, that the mild attacks were due to the introduction of virus attenuated in this unknown manner, when we consider that the bovine species is the one most susceptible to the disease and forms the best soil for the growth and development of the virus, we should have more grounds for supposing that the attack induced by inoculation from the mild case would be more severe in its intensity when introduced into the system of an animal not having the peculiar individual idiosyncracy. However this may be, and whatever view may be taken of the case, it cannot be denied that the results of the methods already tried have been unsatisfactory in the extreme, and their adoption has never been seriously considered in Europe; and in countries in the East where

they have been tried, they have been completely abandoned as not only useless, but harmful, from the fact that they multiplied the centres of contagion.

Up to the present time the recent attempts which have been made in Russia on the one hand by Semmer and in Egypt by Piot have not led to any practical results, and protective inoculation has not yet reached the state of perfection which would permit of its being recommended for general adoption as a means of conferring immunity against rinderpest. That this state may be reached there is every reason to believe, and the results of the investigations which are to be carried out in India are looked forward to with much interest.

Treatment.—The treatment of this disease, as is well known, is seldom followed by very satisfactory results in Europe, whatever the system adopted, and the numbers of recoveries bear but a very small percentage to the numbers of deaths from the disease. This is true also in India as regards the more severe types of the disease, and treatment must always give way to suppression by isolation, segregation, and like measures of preventing spread to other animals in the same and neighbouring herds. Moreover, another point which militates against the adoption of the treatment of cattle in this country is that of expense. If we consider that with treatment the percentage of recoveries is very small comparatively, the small number of recoveries which may be attributed to the action of the drugs administered must bear the expense of the treatment of the whole number; and in cases where the value of the animals is but small, it will be found that the treatment scarcely pays. There are cases, however, where treatment is of great value, when the disease is of a mild type for instance, and there is reasonable hope that the number of recoveries will be large; for we must not lose sight of the fact that an animal recovered is proof against the disease probably for the rest of its life, and therefore a great acquisition in a country where the disease is enzootic. Under favourable circumstances, then, we may adopt such treatment as may seem to us from the pathology of the disease to be most suitable, always bearing in mind that it is necessary, if satisfactory results are to be obtained, that it must be as inexpensive and the drugs used to be obtained in the bazaars of the towns or villages where the disease is prevalent.

When we consider that the more important lesions have their seat in the digestive tract, and that the disease is of a typhoid or typhus nature, we have something at least to guide us in the treatment. The first point to be attended to is the state of the digestive system, and the next is to support the strength of the animal and to limit as much as may be the lowering diarrhœa and dysentery which weaken and destroy the animal.

In the first stage, where the changes in the abomasum and intestine have led to constipation, the bowel being in a more or less inert

condition, it is generally considered advisable to administer a laxative in order to clear out the organs of all the semi-digested food, which, as we are aware, if allowed to remain, forms toxic materials which, when absorbed into the system, give rise to serious disturbance and aggravate the disease, and moreover irritate the digestive mucous membranes with which they remain in contact. This, then, appears to be logically reasonable, and we may use any laxative which it may seem to us most desirable, taking care that its action be not too pronounced or the nature of the laxative too irritant. Veterinary Surgeon Thacker, who has treated some 2,541 cases of the disease with great success, always used for this purpose two or three ounces of finely-divided sulphur in two quarts of gruel and administered morning and evening until some slight laxative effect is observed. The report of the Indian Cattle Plague Commission shows that they agree in this particular, and states that "mild laxatives in the early stages are undoubtedly advantageous, but that severe purgation is positively harmful." Admitting this first step in treatment, then, and still bearing in mind the typhoid nature of the disease, and the lesions found in the digestive tract, the importance of withholding solid food will be evident, considering the inability to digest it which must result from the state of the absorbent mucous membrane, and the consequent irritation which must result from its introduction into the canal. Too much stress, then, can scarcely be laid upon the importance of withholding solid food and substituting for it fluid and easily-digested nutriment; milk and eggs, to which has been added some papaiya fruit, would be the most suitable diet were it not that in many cases papaiya cannot be obtained and milk and eggs are too expensive a form of diet for animals of little value. We have, then, to turn to the next best fluid food, which is gruel of various kinds. The Indian Cattle Plague Commissioners recommend thin rice-water.

When the stage of diarrhœa comes on, or before it rather, we may use with much benefit Veterinary Surgeon Thacker's mixture, which has in my hands been in some cases attended with good results:—

Camphor	2 drs.
Datura	2 „
Chiretta	1 dr.
Arrack	4 ounces

added to two quarts of gruel given morning and evening.

When diarrhœa comes on it is not generally advisable to try to stop it immediately, but rather to allow a little time for some of the virus to be thrown off by this means; but at the same time our best endeavours should be used to prevent, if possible, its running on too long and debilitating the animal too much and thus impeding recovery, and so, after it has existed for about 24 hours, Mr. Thacker and others recommend that opium 1 dr. and catechu 3 drs. be given. Veterinary Surgeon

Marriott found very good results follow the use of opium in large doses in this disease; and I have used a compound of hyoscyamus and opium with good results, but it is too expensive for general use. If the diarrhœa and dysentery progress, we may with benefit supplement the above medicines with decoction of baël fruit, which, as is well known, is almost a specific in these dysenteric diseases. Veterinary Surgeon Gunn recommends the following method of preparing this decoction:—Take 4 seers (8lbs.) of baël fruit, break them up and boil in 12 seers of water; strain whilst hot through a coarse piece of cloth, and place the liquid in a cool place. Give one seer of this fluid every three hours, so long as the diarrhœa and dysentery last. If this be very persistent, carbolic acid may be tried.

With regard to the skin eruption, the pustules should be washed and some mixture or liniment be applied to keep off the flies.

During the whole course of the treatment, the strength must be maintained by gruel and alcoholic stimulants carefully administered. It will be seen from the above that treatment is a troublesome matter, entailing a considerable amount of labour; but in certain cases, as in those treated by Veterinary Surgeon Thacker, who carefully watched and treated through the different stages of the disease 2,541 cases, of which 1,763 recovered, the labour was amply repaid. It appears from the report of the Indian Cattle Plague Commission that it was their opinion that 20 per cent. of animals which would otherwise have died may be saved by treatment.

Prevention and suppression.—It has already been observed that in the present state of our knowledge, the limitation of this disease by protective inoculation cannot be seriously considered, but at the same time it is to be hoped that in time the virus may be by some means attenuated to such a degree as to become a harmless protective medium conferring immunity. Protection therefore being out of the question, the whole of our attention and energy should be given to repression and prevention of the spread of the disease from village to village or from house to house. Nothing can be more deplorable than to watch this malady spreading from village to village, and carrying off numbers of cattle, without any effort being made to stop it, when a little exertion on the part of those in authority would easily limit its ravages. The conditions under which cattle exist in this country are so varied that it is impossible for us to lay down any fixed plan of action, as what would be applicable to one locality would not work in another, so that it must be left to the intelligence of those who are entrusted with the duty of repression. The difficulties which are encountered in certain parts in the matter of the segregation of animals in the same village are so great as to be almost insurmountable, and more especially is this the case where the villages are small.

The insidious character of the invasion of the malady renders it almost certain, generally in small villages, that the majority of cattle in the same village will have been exposed to contagion, owing to the fact that the animals are herded together during non-working hours, feed over the same pastures, drink at the same water, and associate freely on roads and in the fields during working hours. When we take into consideration the fact that the disease is present in an animal for a very considerable time before any marked symptoms of illness are manifested, during which period, if reliance is to be placed on Semmer's experiments, it is capable of communicating the disease to other animals, and that it is soiling the roads and pastures, as also the water, with its saliva and excretions, it is not to be wondered at that large numbers of cattle are attacked apparently in a short time. In addition to this, there is the fact that until some few animals are affected and the disease has reached an advanced stage, it is not recognized, and during the whole of this time so long as they can walk they are driven to graze with the rest of the herd. In villages, therefore, where the whole of the cattle are grazed in one herd, the chance of doing very much by isolation of the sick is small. But there are many cases where several herds are formed and frequent different pastures and watering-places, and are not associated together, and it is in these cases that benefit arises from the adoption of this measure..

With regard to the best and most suitable methods of suppression, it will be best to leave these for the various officers of the Civil Veterinary Department to report upon, as so much depends upon local conditions, customs, and prejudices. This will therefore be done.

G. I. C. P. O.—No. 18 I. G. C. V. D.—23-7-94.—200.